Original Research

Fracture Epidemiology and Control in a Developmental Center

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During 3.5 years, 182 fractures occurred among 994 residents of a developmental center. The fracture rate was 5.2 per 100 person-years (1.7 times greater than the rate in the US population). Fracture rate was significantly greater in residents with: epilepsy, older age, male gender, white race, independent ambulation, osteoporosis, and residence in intermediate care (versus skilled nursing) units; it was not affected by severity of mental retardation. Hand and foot bones were fractured in 58% of cases. Femur fracture occurred in 13 cases (7%). Fracture was caused by a fall in 41 cases (23%); its cause was indeterminable in 105 cases (58%). Fractures, occurring without significant injury, may be an important cause of preventable disability in this population. Control measures are suggested.

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In 1997, there were approximately 146,517 people with developmental disabilities (DD) in California; 4,102 of them resided in a developmental center.[1] To maintain the quality of life for individuals with DD, it is important to prevent acquisition of new disabilities. Attention should therefore be focused on fractures because they can cause substantial morbidity, and many can be readily prevented. Per US data, an average fracture requires active medical care in 97% of cases, and leads to 53 restricted-activity days, 16 bed days, 33 work-loss days, and 8 school-loss days.[2]

Fractures appear to be common in individuals with DD, especially in those with epilepsy.[3–9] To reduce fracture-related disability, it is crucial to identify factors that contribute to this predisposition. Only one study has been published describing the epidemiology of fractures in people with DD.[2] That study, however, was biased, as 98% of its subjects had epilepsy, and were therefore prone to fractures.[3–9] We present the epidemiology of fractures in a center whose residents are more representative of the general population of people with DD, the majority of whom have no epilepsy.

Method

This study was conducted at Fairview Developmental Center, one of five long-term care facilities in California for people with DD. Its features have been described previously.[10] Consistent with a national trend for community care of people with lesser DD,[11] and the Coffelt settlement requiring the same,[12] the Fairview Developmental Center resident census has decreased to 40% of its licensed capacity since its dedication in 1961. Consequently, current residents are older, with more severe medical or behavioral impairments.[11]

Licensed staff routinely monitor all residents for injury (pain, swelling, bruising, refusal to use a part). All suspected injuries are evaluated by a physician and radiographed, then a special incident report is prepared in compliance with state regulation. All fractures are listed on a fracture log. We reviewed records of all persons who had a fracture during the preceding 3.5 years.

For all residents, information was obtained about age, gender, race, level of mental retardation, care level (skilled nursing or intermediate care), mobility (ability to move about independently with or without a wheelchair), clinical diagnosis of osteoporosis or epilepsy, and cause of fracture. If a single injury fractured multiple bones, each fracture was counted separately. No resident received alendronate or electroconvulsive therapy. Common anticonvulsants prescribed for epileptics were phenobarbital, phenytoin, carbamazepine, valproic acid, and gabapentine.

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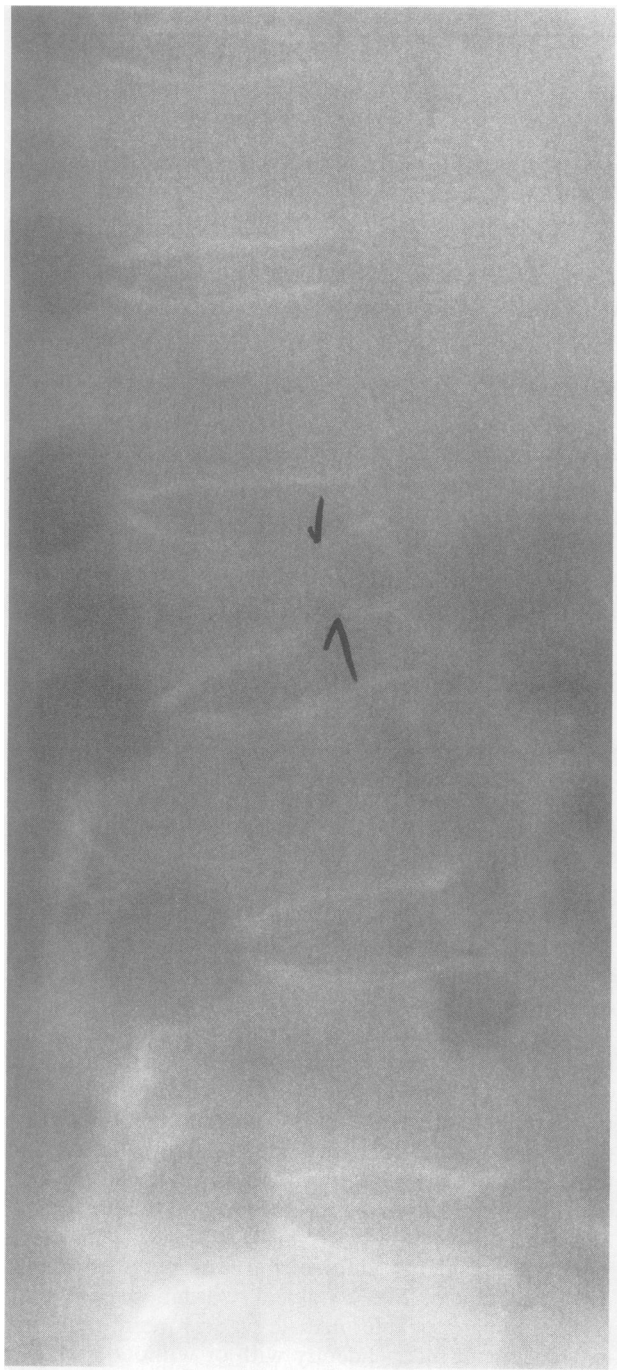

Figure 1.—Radiograph illustrating osteoporosis. The bold paired arrows point to a compression fracture of a vertebral body. There is loss of trabecular bone causing accentuation of the vertebral end plates (curved arrow). Notice the loss of normal contrast between the radiodensity of spinal column and adjacent soft tissues, reflecting reduced bore mass (porosis: porous bone).

Observed fracture rates were standardized against the US population rates for age, sex, and race (Table 1). One-sided Poisson probability test was used to determine the significance of the difference between observed and expected rates using a Bonferroni corrected standard. Poisson probability value of <.01 was considered statistically significant. For features with no national rates, relative risk was estimated by determining the odds ratio and 95% confidence limits. The odds ratio was considered significant at $P < .05$ if the 95% confidence bounds did not incorporate the number 1.[13]

Results

The Center's midstudy census was 994 residents. One or more fractures occurred among 114 (11.5%) residents. During 3.5 years, 182 bones were fractured, yielding a fracture rate of 5.2 fractures per 100 person-years (Table 1). The observed rate was 1.7 times greater than the fracture rate (3.0 fractures per 100 person-years) for the US population[2].

Fracture rates standardized for age, sex, and race are presented in Table 1. The observed number of fractures (n = 182) was significantly greater than the expected number of fractures (n = 104). The fracture rate was significantly greater in residents with epilepsy, male gender, older age, white race, osteoporosis, ambulation, and residence in intermediate-care-units. Rate was not affected by the severity of mental retardation (Table 2).

Fracture incidence stratified by the affected bone is presented in Table 3. Most fractures (n = 105 or 58%) occurred in hand (n = 62) and foot (n = 43) bones. Fracture incidence according to the number of fractures per affected subject is presented in Table 4; 87% of subjects sustained only one or two fractures. Causes of fractures are presented in Table 5; falls were related to 41 (23%) fractures. Features suggestive of osteoporosis (Figure 1) and osteomalacia (Figure 2) were present in occasional radiographic films.

Discussion

The fracture rate is significantly greater in our residents than the noninstitutionalized civilian US population (5.2 versus 3.0 fractures per 100 person-years, Table 1).[2] This is a cause for concern. Most civilians sustain fractures following significant trauma related to work, sports, or automobiles. In contrast, our residents are at minimal risk for such injury as they live in a sheltered environment. Fewer than 10% are employed (in low hazard occupations such as collating, sorting, assembling, and food serving). Sports are light, and involve minimal physical contact. Road trips are few, so no commute is involved.

It can be argued that the published fracture incidence for the US population is spuriously low due to poor case finding related to the use of interviews as the data source. This potential weakness is unlikely to be significant, since 97% of all fractures inevitably produce clinical manifestations leading to a definite diagnosis. Contrarily, the US rate may be spuriously high as its numerator, but not ours, includes dislocations in addition to fractures.[2]

TABLE 1.—*Observed Fractures Standardized for Age, Sex, and Race with US Population*

Feature, Number of Residents (n)	*Number of Fractures Observed/Expected in 3.5 y*	*Rate Observed/Expected per 100 Person-y*	*Poisson Probability One-sided Test*
Males, age			
<5 y, *n* = 3	0/0.1	0/1.5	N/A
5–17 y, *n* = 21	3/3.8	4.1/5.1	.72268
18–44 y, *n* = 458	91/64.1	5.7/4	.00090
>45 y, *n* = 97	20/8.2	5.9/2.4	.00032
Total, *n* = 579	114/76	5.6/3.6	.00001
Females, age			
<5 y, *n* = 2	0/.2	0/2.4	N/A
5–17 y, *n* = 21	1/3	1.4/4.1	.95088
18–44 y, *n* = 309	40/16.2	3.7/1.5	.00001
>45 y, *n* = 83	27/9	9.3/3.1	.00001
Total, *n* = 415	68/28	4.7/2.5	.00001
Race			
White, *n* = 771	165/89	6.1/3.3	.00001
Nonwhite, *n* = 223	17/17	2.2/2.2	.54864
Total, *n* = 994	182/104	5.2/3.0	.00001

Poisson probability: The difference between the observed rate and the expected rate is statistically significant if the value is less than .01, using a Bonferroni corrected standard of .0009 = .01/11.
N/A: Not Applicable. Sample size and/or number of observations are insufficient to test the hypothesis.
Calculation of observed fracture rate per 100 person years: Number of fractures in 3.5 years × 100 ÷ resident census ÷ 3.5 years.

TABLE 2.—*Epidemiologic Features of Study Population, Number of Bones Fractured, and Fracture Rates During 3.5 Years*

Feature	*Sample Size, n*	*Fractures: Number, Rate per 100 Person-y*	*Odds Ratio*	*95% Confidence Bounds*
Epilepsy			1.886	1.356–2.624
Present	295	75, 7.3		
Absent	699	107, 4.4		
Gender			2.088	1.337–3.261
Male	579	114, 5.6		
Female	415	68, 4.7		
Race			3.2999	2.002–5.438
White	771	165 6.1		
Nonwhite	223	17 2.2		
Osteoporosis			2.794	1.973–3.956
Present	196	64, 9.3		
Absent	798	118, 3.0		
Mobility			.329	.218–.497
Nonambulatory	317	28, 2.5		
Ambulatory	677	154, 6.5		
Care level			.426	.300–.607
Skilled nursing	412	47, 3.3		
Intermediate care	582	135, 6.6		
Mental retardation			.967	.662–1.413
Profound	764	139, 5.2		
Nonprofound	230	43, 5.3		

Odds ratio is an estimate of the relative risk. The observed difference is significant at $P < .05$ if the 95% confidence bounds do not incorporate the number 1.
Note: fracture rate for US population not available for these groups.

TABLE 3.—*Fracture Incidence According to the Affected Bone*

Bone name	*Number*	*%*
Skull	1	0.5
Vertebra	2	1.1
Nose	7	3.8
Face	3	1.6
Clavicle	4	2.2
Humerus	7	3.8
Radius	3	1.6
Ulna	3	1.6
Carpal bones	0	0
Metacarpal bones	19	10.4
Phalanx, hand	43	23.6
Rib	10	5.5
Sternum	0	0
Pelvis	0	0
Femur	13	7.1
Patella	1	0.5
Tibia	9	4.9
Fibula	11	6.0
Tarsal bones	3	1.6
Metatarsal bones	19	10.4
Phalanx, foot	24	13.2
Total fractures	182	100.0

TABLE 4.—*Frequency of Fractures per Resident During 3.5 Years*

Fractures per Resident	*Number of Residents*	*Total Fractures*
1	69	69
2	30	60
3	9	27
4	5	20
5	0	0
6	1	6
Grand total	114	182

TABLE 5.—*Fractures Stratified by their Causes*

Cause of Fracture	*Fractures*	*Number %*
Fall		
From wheelchair	4	2
From bed	3	2
Spontaneous/seizure	34	19
Assault by peer	5	3
Self-mutilation	11	6
Accident*	15	8
Care-related†	5	3
Unknown	105	58
Total	182	100

*Accidents: caught in door, 8; falling objects, 7.
†Care-related: transfers from bed to chair, therapeutic exercises.

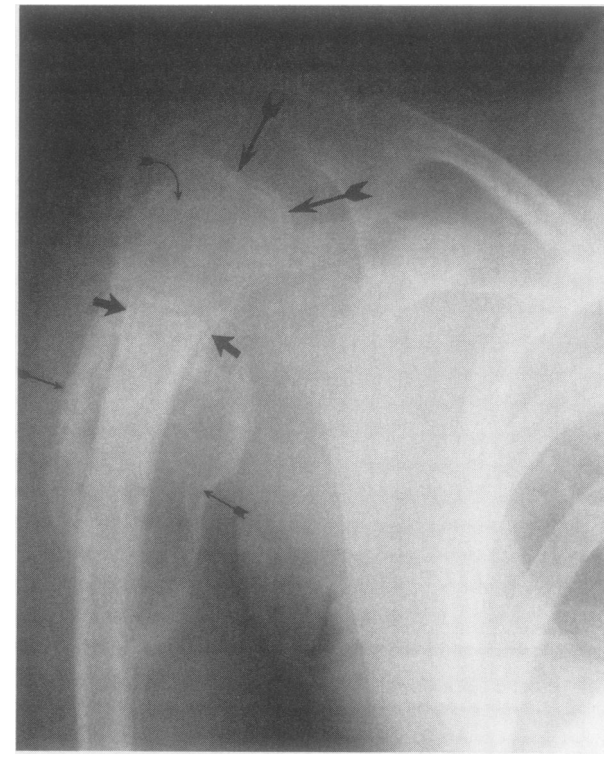

Figure 2.—Radiograph illustrating osteomalacia. The two short bold arrows point to a healing fracture of the humerus with abundant callus formation (paired thin arrows). Curved arrow points to the prominent bone trabeculae. Note the delay in fusion of the proximal humeral epiphysis (two bold serrated arrows) with the shaft in this 33-year-old man; fusion usually is complete by the age of 18 years. Note the abnormal curvature of the humeral shaft reflecting bone softening (malacia).

The fracture rate was significantly higher among older residents (Table 1). This may indicate that the older residents represent a different cohort of subjects. The younger group has had lesser exposure to psychotropic medicines (20% in 1999 versus 60% in 1977), and has received more balanced nutrition (meeting US Recommended Daily Allowance, planned by licensed dietitians), and physical and occupational therapy. Higher fracture rate among the older residents may also reflect the combined effect of aging, immobilization-osteoporosis, and anticonvulsant-osteomalacia.

Of all residents, 295 (30%) had epilepsy requiring anticonvulsants. The fracture rate was higher among epileptics (Table 2), as observed previously.[3–9] This is probably due to fractures caused by violent muscle contractions, atonic falls, osteoporosis, and osteomalacia. Residents with drug-refractory seizures may develop immobilization-osteoporosis due to activity restrictions, imposed because financial constraints restrict the provision of one-on-one supervision.[14] Anticonvulsants (phenobarbital, carbamazepine, and phenytoin) may cause osteomalacia, as they induce liver microsomal enzymes

TABLE 6.—*Recommendations for Fracture Control at the Developmental Center*

I. Primary prevention: Reduce fracture incidence.

1. Host Factors: Interdisciplinary team should assess each resident's risk for fractures and develop a comprehensive customized fracture prevention plan. Consider history of prior fractures; risk for falls (seizure, prior fall, dizziness, cognitive impairment, postural hypotension, drug side effect, cardiac arrhythmia, impaired mobility); seizure (type, frequency, aura, prior injury); osteoporosis (immobility, age, calcium and estrogen supplement); and osteomalacia (sun exposure, vitamin D intake, anticonvulsants). Screen fracture-risk residents for osteoporosis by noninvasive dual energy x-ray absorptiometry,[20] or bone turnover markers (serum osteocalcin, urine n-telopeptide), and for osteomalacia by serum and/or urine levels of calcium, phosphorus, and 25-hydroxy vitamin D.
2. Enhanced bone strength: Maintain good nutrition. Prevent anticonvulsant-osteomalacia by calcium and vitamin D supplements and sunlight exposure. Offer physiotherapeutic and swimming exercises starting at an early age to maintain bone mass. Prevent and treat osteoporosis with alendronate,[21] and estrogen replacement therapy for females.
3. Adequate diagnosis and treatment of epilepsy: Preferential use of anticonvulsant monotherapy.
4. Personal protective devices: Facilitate normalization. Avoid custodial mentality. Use helmets and restraints as the last resort. Ensure proper helmet fit. Use a chin strap to prevent inadvertent removal by the resident or unintentional stripping during a fall. Use padded bed-rails for residents prone to a seizure or fall.
5. Injury prevention: Provide adequate staffing with trained and caring personnel. Provide individualized supervision through foster grandparents. Provide assistance during ambulation. Use proper lifting techniques and equipment for assisted transfers. Effectively manage residents with maladaptive behavior. Provide well-fitting footwear. Avoid loose shoe laces or excessively long pants. Develop good vocational programs. Provide more outdoor and recreational areas to keep residents occupied.
6. Safe Environment: Provide adequate illumination. Eliminate hazards (obstacles, throw rugs, floor wires, stray toys, wet or slippery floors). Repair doors that close abruptly to prevent fingers getting caught between door and the door jam. Install nonslip floorings, handrails. Fill gopher holes in ground. Use low beds for persons prone to fall from bed. Use shower chairs and padded pedestal baths as indicated. Provide separate accommodation for fracture-prone and blind residents away from wheelchair-users or those with maladaptive behavior. Avoid excessive noise and traffic in hallways. Evaluate resident density and space per person.
7. Wheelchair safety: Provide residents their usual wheelchairs. Install spoke covers for self-propelling residents, anti-tip devices, padded side rails, foot rests, etc.

II. Secondary prevention: Reduce fracture morbidity by early diagnosis and treatment.

1. High index of suspicion, careful physical examination, prudent use of radiographs.
2. Prompt and intensive treatment of all fractures.

III. Tertiary prevention: Reduce fracture-related morbidity.

1. Physical medicine and rehabilitation: Correction of deformities and use of orthotic devices to aid in ambulation.
2. Corrective orthopedic management of fracture malunions and nonunions.

to inactivate vitamin D and produce hypovitaminosis D, inhibit intestinal calcium transport, cause secondary hyperparathyroidism, and suppress osteoblasts.[15,16]

Inexplicably, the fracture rate was higher in the male gender. Higher fracture rate among white subjects probably reflects their greater predisposition to osteoporosis.[14] The fracture rate was not affected by the level of mental retardation.

The fracture rate was higher among residents with osteoporosis (Table 2). Persons of all ages, who present with fractures sustained from minimal trauma without other bone disease, probably have osteoporosis.[14,17,18] The cause of osteoporosis in our residents is probably prolonged immobilization related to severe neurologic-orthopedic disability.[14] This theory is corroborated by the fact that immobilization-osteoporosis leads to a balanced decrease in cortical and trabecular bone mass, and results in fractures involving all bones,[18] as noted in our study (Table 3). In contrast, the commoner postmenopausal osteoporosis predominantly affects vertebrae (trabecular bone).

The fracture rate was higher among ambulatory residents (Table 2). This is probably secondary to their increased physical activity, or falls (due to seizure, unsteady gait, drug side effects, maladaptive behavior, and orthopedic or neurologic disability). Probably for the same reasons, the fracture rate was higher among residents requiring intermediate care (usually ambulatory) than residents receiving skilled nursing care (who tend to be more disabled, sicker, or bedridden). The lower fracture rate in bedridden residents may be counterintuitive, as recumbency would seem to predispose them to osteoporosis. This risk, however, may be offset by their reduced risk for injuries and superb handling by caring staff.

Femur fractures occurred in 13 subjects (7%) (Table 3). These are among the most clinically significant fractures, as they generally require surgical fixation and extended convalescence, and predispose patients to complications (eg, impaired ambulation, pneumonia, pressure ulcers, urinary tract infection, and reduced life span).[14]

Fractures of hand (n = 62) and foot (n = 43) bones accounted for 105 (58%) of the 182 fractures (Table 3). Although these small bone fractures usually require only conservative management, they can nonetheless considerably interfere with activities of daily living in persons with preexisting handicaps. Small bones are probably at a higher risk of injury because of greater interaction with the environment (for example, during opening or closing of doors), defense against injuries (bracing with hands

and feet to prevent a fall, or self-defense against assault), and relative underprotection against extraneous forces (due to their exposed location) compared to the trunk.

Vertebral fracture was recorded in only 2 cases (1.1%) (Table 3). Its low incidence is puzzling, as it is reportedly common in persons with epilepsy[19] or osteoporosis.[14] In this study, vertebral fracture was probably underdiagnosed due to the lack of a universally accepted radiological definition, difficulty in diagnosis in persons with coexisting severe spinal deformities, its frequent asymptomatic nature[14] (especially in bedridden nonverbal patients), and potential underdiagnosis due to the retrospective nature of our study.

Of the 114 subjects who sustained a fracture, 45 (40%) had a repeat fracture accounting for a total of 113 (62% of all) fractures (Table 4). This suggests that persons with a prior fracture are at an increased risk of future fractures. Predisposition to recurrent fractures was rather dramatic in a young man with epilepsy and osteoporosis who sustained a total of 18 fractures during 19 years.[9]

The cause of fracture was unknown in 105 cases (58%) (Table 5). Due to the severity of their mental retardation, many residents are unable to identify an injury or verbalize its effects. Many fractures cannot be diagnosed unless there is apparent deformity, bruise, impairment of motion, or refusal to use a part. Since many subjects are nonambulatory, fracture detection may be even more difficult. Since minor injuries (due to fall, assault, seizure, head banging, temper tantrum, or during transfer from bed to chair) are frequent, it may be difficult to attribute a given fracture to a specific injury. Most fractures of unknown cause were probably related to an unwitnessed seizure or fall.

Comparing our findings with two other studies of people with DD is instructive. In New England, during 10 months (June 1986–April 1987), 61 fractures occurred among 55 residents (rate: 13.2 fractures per 100 person-years).[3] This higher fracture rate may be because 98% of the subjects had epilepsy compared to only 30% in our study, and may also reflect better case finding in a prospective study. No vertebral fracture was recorded; 52% of fractures involved hand and foot bones. The femur was fractured in 6 cases (10%). The cause of fractures was known in 64% of cases (compared to 42% in our study); this greater knowledge may be related to the prospective nature of their study.

In our sister developmental center in Northern California, during 2 years (February 1995–January 1997), 104 fractures occurred among 961 residents (rate: 5.4 fractures per 100 person-years) (unpublished report; Fractures at Sonoma Developmental Center, 1995–1997; Iacovoni V, Jenkins D, Phillips S, May 1, 1997). No vertebral fracture occurred; 55% of the fractures involved hand and foot bones. The femur was fractured in 12 cases (12%). Fracture cause was known in only 30% of cases. These findings are similar to ours and reflect similarity of our two populations.

Our study suggests that fractures are an important cause of preventable morbidity in people with DD. It behooves their caretakers and physicians to exercise extra care not to let a fracture go undiagnosed. Pain, particularly post-seizure pain, should not be dismissed as a musculoligamentous injury until a fracture has been excluded radiographically. Diagnosis may be difficult due to lack of a history of significant trauma, difficulty in examining a person with severe contractures, and absence of additional functional impairment in an already nonambulatory nonverbal person.

In a nondisabled person, a fracture usually resolves with no permanent disability. In a disabled individual with marginal mental and physical reserves, however, it can produce additive, and at times, multiplicative permanent disability. The outcome may be devastating to that resident's independence, developmental programming, or activities of daily living. Although preventive health care is important for all, it can provide a much greater proportional return in people with DD. With major recent advances in the diagnosis and treatment of osteoporosis, a lot more can be done to control fractures than was possible a decade ago.[20,21] Steps recommended to prevent fractures at our center are listed in Table 6. Continued surveillance and follow-up studies are necessary to evaluate the effectiveness of these control measures.

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